PB88-173638

Health and Environmental Effects Profile for Dinitrobenzenes

(U.S.) Environmental Protection Agency
Cincinnati, OH

Sep 85

U.S. Department of Commerce
National Technical Information Service
NTIS

TECHNICAL REPORT DATA
(Please read Instructions on the reverse before completing)

1. REPORT NO.	2.	3. RECIPIENT'S ACCESSION NO.
EPA/600/X-85/361		PB88-173638

4. TITLE AND SUBTITLE	5. REPORT DATE
Health and Environmental Effects Profile for Dinitrobenzenes	September 1985
	6. PERFORMING ORGANIZATION CODE
7. AUTHOR(S)	8. PERFORMING ORGANIZATION REPORT NO.
9. PERFORMING ORGANIZATION NAME AND ADDRESS	10. PROGRAM ELEMENT NO.
	11. CONTRACT/GRANT NO.
12. SPONSORING AGENCY NAME AND ADDRESS Environmental Criteria and Assessment Office Office of Research and Development U.S. Environmental Protection Agency Cincinnati, OH 45268	13. TYPE OF REPORT AND PERIOD COVERED
	14. SPONSORING AGENCY CODE EPA/600/22

15. SUPPLEMENTARY NOTES

16. ABSTRACT

The Health and Environmental Effects Profile for dinitrobenzenes was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA) and to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Dinitrobenzenes has been determined to be a systemic toxicant. An Acceptable Daily Intake (ADI), defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect, for dinitrobenzenes is 0.028 mg/day for oral exposure.

The Reportable Quantity (RQ) value of 1, 10, 100, 1000 or 5000 pounds is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified by CERCLA based on chronic toxicity. The RQ value for dinitrobenzenes is 100.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group

18. DISTRIBUTION STATEMENT	19. SECURITY CLASS (This Report)	21. NO. OF PAGES
Public	Unclassified	61
	20. SECURITY CLASS (This page)	22. PRICE
	Unclassified	A04 14.95

EPA Form 2220-1 (Rev. 4-77) PREVIOUS EDITION IS OBSOLETE

EPA/600/X-85/361
September 1985

PB88-173638

HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR DINITROBENZENES

ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
OFFICE OF HEALTH AND ENVIRONMENTAL ASSESSMENT
OFFICE OF RESEARCH AND DEVELOPMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
CINCINNATI, OH 45268

REPRODUCED BY
U.S. DEPARTMENT OF COMMERCE
NATIONAL TECHNICAL
INFORMATION SERVICE
SPRINGFIELD, VA. 22161

DISCLAIMER

This document has been reviewed in accordance with the U.S. Environmental Protection Agency's peer and administrative review policies and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste and Emergency Response by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA), as well as to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched." The literature search material is current through November, 1984.

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor of q_1^* (U.S. EPA, 1980) is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

Reportable quantities (RQs) based on both chronic toxicity and carcinogenicity are derived. The RQ is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified under CERCLA. These two RQs (chronic toxicity and carcinogenicity) represent two of six scores developed (the remaining four reflect ignitability, reactivity, aquatic toxicity and acute mammalian toxicity).

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA and CERCLA dockets.

TABLE OF CONTENTS

		Page
1.	INTRODUCTION	1
1.1.	STRUCTURE AND CAS NUMBER	1
1.2.	PHYSICAL AND CHEMICAL PROPERTIES	1
1.3.	PRODUCTION FILE	3
1.4.	USE DATA	5
2.	ENVIRONMENTAL FATE AND TRANSPORT PROCESSES	6
2.1.	WATER	6
2.1.1.	Hydrolysis	6
2.1.2.	Oxidation	6
2.1.3.	Photolysis	6
2.1.4.	Microbial Degradation	7
2.1.5.	Transport	8
2.2.	AIR	10
2.3.	SOIL	10
2.3.1.	Microbial Degradation	10
2.3.2.	Chemical Degradation	11
2.3.3.	Photolysis	11
2.3.4.	Adsorption/Leaching	11
2.4.	SUMMARY	12
3.	EXPOSURE	13
3.1.	WATER	13
3.2.	FOOD	13
3.3.	INHALATION	14
3.4.	DERMAL	14
4.	PHARMACOKINETICS	15
4.1.	ABSORPTION	15
4.2.	DISTRIBUTION	15
4.3.	METABOLISM	15
4.4.	EXCRETION	19
5.	EFFECTS	20
5.1.	CARCINOGENICITY	20
5.2.	MUTAGENICITY	20
5.3.	TERATOGENICITY	22
5.4.	OTHER REPRODUCTIVE EFFECTS	22
5.5.	CHRONIC AND SUBCHRONIC TOXICITY	22
5.6.	OTHER RELEVANT INFORMATION	26

TABLE OF CONTENTS (cont.)

Page

6. AQUATIC TOXICITY. 29

6.1. ACUTE EFFECTS. 29
6.2. CHRONIC EFFECTS. 30
6.3. PLANT EFFECTS. 30
6.4. RESIDUE. 31
6.5. OTHER REVELANT INFORMATION 31

7. EXISTING GUIDELINES AND STANDARDS 32

7.1. HUMAN. 32
7.2. AQUATIC. 32

8. RISK ASSESSMENT 33

9. REPORTABLE QUANTITIES 35

9.1. REPORTABLE QUANTITY (RQ) RANKING BASED ON CHRONIC TOXICITY 35
9.2. WEIGHT OF EVIDENCE AND POTENCY FACTOR ($F=1/ED_{10}$) FOR CARCINOGENICITY. 37

10. REFERENCES. 39

APPENDIX: LITERATURE SEARCHED. 51

LIST OF TABLES

Table No.	Title	Page
1-1	Structures, Molecular Weights, CAS Numbers and Empirical Formulas of the Dinitrobenzenes.	2
1-2	Dinitrobenzene Production Data from the U.S. EPA TSCA Production File.	4
4-1	Metabolities of 1,3-Dinitro[^{14}C]benzene Recovered from the Urine and Feces of Rabbits 2 Days After Oral Administration Expressd as Percentage of Administered Radioacticity .	16
5-1	Mutagenicity of 1,3-Dinitrobenzene	21
5-2	The Average Daily Intake of 1,3-Dinitrobenzene in Rats	23
5-3	Acute Toxicity of 1,3-Dinitrobenzene in Rats	27
9-1	Composite Scores for the Oral Toxicity of 1,3-Dinitrobenzene in Rats	36
9-2	1,3-Dinitrobenzene: Minimum Effective Dose (MED) and Reportable Quantity (RQ)	38

LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
BCF	Bioconcentration factor
bw	Body weight
CAS	Chemical Abstracts Service
CNS	Central nervous system
COD	Chemical oxygen demand
CS	Composite score
DNA	Deoxyribonucleic acid
EC_{50}	Effective concentration to 50% of recipients
K_{oc}	Soil adsorption coefficient
K_{ow}	Octanol/water partition coefficient
LC_{50}	Concentration lethal to 50% of recipients
LD_{50}	Dose lethal to 50% of recipients
LT_{20}	Exposure time for 20% mortality
MED	Minimum effective dose
NOAEL	No-observed-adverse-effect level
NOEC	No-observed-effect concentration
ppm	Parts per million
RQ	Reportable quantity
RV_d	Dose-rating value
RV_e	Effect-rating value
STEL	Short-term exposure level
TCDD	Tetrachlorodibenzo-p-dioxin
TLV	Threshold limit value
TNT	Trinitrotoluene

LIST OF ABBREVIATIONS (cont.)

TWA	Time-weighted average
UV	Ultraviolet
WS	Water solubility

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

The structures, CAS Registry numbers, molecular weights and empirical formulas of the dinitrobenzenes are presented in Table 1-1.

1.2. PHYSICAL AND CHEMICAL PROPERTIES

The dinitrobenzenes are colorless to yellowish crystalline solids at room temperature (Verschueren, 1983). Selected available physical properties of 1,2-dinitrobenzene, 1,3-dinitrobenzene, and 1,4-dinitrobenzene are given below:

1,2-Dinitrobenzene

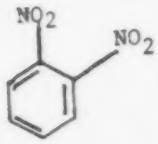
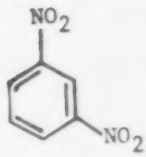
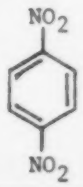
Melting point:	118°C	Verschueren, 1983
Boiling point:	319°C (at 773 mm Hg)	Verschueren, 1983
Specific gravity:	1.565 (17/4°C)	Verschueren, 1983
Water solubility:		
at 25°C	2100 ppm	U.S. EPA/NIH, 1984
at 100°C	3800 ppm	Verschueren, 1983
Conversion factor (air):	1 mg/m³ = 0.14 ppm	Verschueren, 1983
Log K_{ow}:	1.58	Hansch and Leo, 1981

1,3-Dinitrobenzene

Melting point:	89.8°C	Verschueren, 1983
Boiling point:	300-302°C (770 mm Hg)	Verschueren, 1983
Specific gravity:	1.571 (0/4°C)	Verschueren, 1983
Water solubility:		
at 15°C	469 ppm	Verschueren, 1983
at 100°C	3200 ppm	Verschueren, 1983
at 25°C	500 ppm	U.S. EPA/NIH, 1984
at 20°C	0.0022 moles/ℓ	Spanggord et al., 1980

TABLE 1-1

Structures, Molecular Weights, CAS Numbers, and Empirical Formulas of the Dinitrobenzenes*

Compound and Synonyms	CAS Number	Molecular Weight	Empirical Formula	Structure
1,2-Dinitrobenzene o-Dinitrobenzene o-DNB	528-29-0	168.11	$C_6H_4N_2O_4$	
1,3-Dinitrobenzene m-Dinitrobenzene m-DNB	99-65-0	168.11	$C_6H_4N_2O_4$	
1,4-Dinitrobenzene p-Dinitrobenzene p-DNB Dithane	100-25-4	168.11	$C_6H_4N_2O_4$	

*Source: NIOSH, 1979

Vapor pressure: at 20°C	0.0039 mm Hg (extrapolated, supercooled liquid)	Spanggord et al., 1980
Log K_{ow}:	1.49	Hansch and Leo, 1981

1,4-Dinitrobenzene

Melting point:	173°C	Verschueren, 1983
Boiling point:	299°C (777 mm Hg) (sublimes)	Verschueren, 1983
Specific gravity:	1.625 (20/4°C)	Verschueren, 1983
Water solubility: at 100°C at 20°C	 1800 ppm 80 ppm	 Verschueren, 1983 U.S. EPA/NIH, 1984
Log K_{ow}:	1.46-1.49	Hansch and Leo, 1981

1.3. PRODUCTION DATA

Production data available from the U.S. EPA TSCA Production File for 1977 (U.S. EPA, 1977) is listed in Table 1-2. DuPont is listed as the only United States producer of 1,3-dinitrobenzene in 1983 (USITC, 1984). USITC (1984) and SRI (1984) do not list any United States manufacturers of 1,2- or 1,4-dinitrobenzene, and recent production volumes are not available. Production of 1,3-dinitrobenzene in 1972 was reported to be 12 million pounds (U.S. EPA, 1980).

Imports of 1,4-dinitrobenzene through principal United States customs districts in 1982 and 1980 were reported to be 2.6 and 21.7 thousand pounds, respectively, while imports of 1,3-dinitrobenzene were 500 pounds in 1980 (USITC, 1981, 1983).

TABLE 1-2

Dinitrobenzene Production Data from the
U.S. EPA TSCA Production File*

Producer (Location)	Type of Production	Production Range
1,2-Dinitrobenzene	NR	NR
1,3-Dinitrobenzene		
DuPont (Deepwater, NJ)	manufacturer (used on-site only)	confidential
American Hoechst (Bridgewater, NJ)	importer	confidential
1,4-Dinitrobenzene		
Eastman Kodak (Rochester, NY)	manufacturer	none

*Source: U.S. EPA, 1977

NR = Not reported

The dinitrobenzenes are manufactured by the nitration of nitrobenzene (or benzene) with a hot mixture of nitric and sulfuric acids. The meta-isomer is predominately formed during this process (TSCA-ITC, 1984). The dinitrobenzenes are formed in small volume as by-products during the manufacture of nitrobenzene (Dunlap, 1981). 1,3-Dinitrobenzene is also a by-product of the manufacture of TNT explosive at the Radford, Volunteer, Joliet and Newport Army Ammunition Plants (Kitchens et al., 1979); it is an impurity in the final TNT product and in waste discharges.

1.4. USE DATA

Nearly all of the 1,3-dinitrobenzene produced by DuPont is used as an on-site intermediate in the production of m-phenylenediamine, which is used mainly as a dye intermediate (TSCA-ITC, 1984). In addition to being used for this purpose, the dinitrobenzenes can be used in organic syntheses and as a camphor substitute in celluloid production (U.S. EPA/NIH, 1984).

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

Like other chemicals, the fate of the dinitrobenzenes in the environment depends upon their ability to undergo chemical, photochemical and biological alterations and to transport or partition from one environmental matrix into another.

2.1. WATER

2.1.1. Hydrolysis. Aromatic nitro compounds are generally resistant to hydrolysis (Lyman et al., 1982). Spanggord et al. (1980) have speculated that 1,3-dinitrobenzene is not expected to hydrolyze under environmental conditions. The same is probably true for the two other isomers.

2.1.2. Oxidation. Pertinent data regarding the oxidation of dinitrobenzenes in the aquatic environment were not located in the available literature as cited in the Appendix. The chemical structure of these compounds would suggest, however, that aquatic oxidation is not significant (Spanggord et al., 1980).

2.1.3. Photolysis. The UV absorption spectrum of 1,3-dinitrobenzene shows an absorption maximum at 233 nm (Kitchens et al., 1979), but it has some absorption in the >300 nm region (Spanggord et al., 1980); this indicates a potential for direct photolysis. Spanggord et al. (1980) reported the results of a laboratory experiment in which 50% of the 1,3-dinitrobenzene component of a TNT condensate water was lost after exposure to sunlight for ~12 days. A control solution maintained in the shade, however, showed an equal loss of 1,3-dinitrobenzene in ~14 days; therefore, the authors suggested that volatilization is more rapid than direct photolysis under the experimental conditions.

Data regarding the photosensitized photolysis of the dinitrobenzenes in the aquatic environment were not located in the available literature as cited in the Appendix. Additional data are required to assess the relative significance of photolysis in the aquatic fate of the dinitrobenzenes.

2.1.4. **Microbial Degradation.** Hallas and Alexander (1983) examined the biodegradation of 1,2- and 1,3-dinitrobenzene by a primary effluent of raw municipal sewage under both aerobic and anaerobic conditions at 29°C. After 28 days of incubation, degradation of 1,2- and 1,3-dinitrobenzene under aerobic conditions was ~30 and 40%, respectively. Under anaerobic conditions, ~80% of the 1,3-isomer degraded in 14 days. Nitroaniline was identified as a degradation product of both isomers under anaerobic and aerobic conditions.

Mitchell and Dennis (1982) studied the degradation of 1,3-dinitrobenzene in water samples collected from the Tennessee River downstream from an Army munition plant, which discharged wastewaters containing the compound into the river. In nonsterilized water samples, the rate of disappearance of a 5 µg/mℓ concentration reached a maximum following a 10-day lag period and proceeded until the 15th day, after which the compound could not be detected. Following the 10-day lag period, the half-life of 1,3-dinitrobenzene at 25°C and with an assumed bacteria level of 10^6 colony-forming units/mℓ was estimated to be ~1 day. In autoclaved samples of the same water, no degradation was observed for a period of up to 20 days. Different water samples were obtained from two rivers and two streams in Maryland and were similarly tested for 1,3-dinitrobenzene degradation. Although these nonsterilized samples contained approximately equal numbers of organisms as those samples from the Tenessee River, degradation did not occur. The authors suggested that the microorganisms in the Tenessee River samples were

adapted to 1,3-dinitrobenzene due to previous exposure. Enrichment cultures from the Tennessee River were reported to grow moderately on 1,3-dinitrobenzene as a sole carbon source. Naumova et al. (1982) reported that Pseudomonas denitrificans grows moderately on 1,3-dinitrobenzene.

The Japanese MITI test indicated that 1,3-dinitrobenzene is resistant to degradation (Kawasaki, 1980); while Kitano (1978) reported that all three dinitrobenzenes are resistant to degradation in the MITI test. When hydrogen is present, enzyme preparations of Veillonella alkalescens are capable of reducing the nitro groups of all three dinitrobenzenes (McCormick et al., 1976). Pitter (1976) used an activated sludge to test the biological degradability of 1,3- and 1,4-dinitrobenzene but observed zero COD removal after 20 days. Bringmann and Kuehn (1971) observed a >95% removal of 1,3-dinitrobenzene (118-146 ppm) in a two-stage aerated wastewater reactor which contained Azotobacter agilis in the first stage and activated sludge in the second stage. In a degradation study with benzene-acclimated activated sludge, Malaney and McKinney (1966) observed little or no degradation of either 1,3- or 1,4-dinitrobenzene; Tabak et al. (1964) and Chambers et al. (1963) found similar results with phenol-adapted bacteria with respect to 1,3-dinitrobenzene. Cartwright and Cain (1959) reported that an extract of Nocardia erythropolis reduced 1,3-dinitrobenzene to the corresponding arylamine.

2.1.5. Transport.

2.1.5.1. **VOLATILIZATION** -- Smith et al. (1981) measured the volatilization rate of 1,3-dinitrobenzene from aqueous solution (10 ppm) in laboratory glassware at room temperature. The solution was subjected to varying speeds of mechanical stirring and varying air flows from an electrical fan.

In solution depths ranging from 7.5-15.5 cm, the volatilization rate constant was found to range from 0.001-0.0031 hr^{-1}; these translate into half-lives of 9-29 days. Therefore, 1,3-dinitrobenzene may be classified as a relatively low volatility compound. Smith et al. (1981) also attempted to measure the Henry's Law Constant but encountered experimental difficulties in part due to slow loss of 1,3-dinitrobenzene from solution. Based on the physical properties of 1,3-dinitrobenzene, a Henry's Law Constant of 2.5×10^{-7} atm-m^3/mol (0.19 torr M^{-1}) was assigned to 1,3-dinitrobenzene at 25°C; however, Smith et al. (1981) noted that the value could also be calculated to be at least one order of magnitude greater. Using the water solubility and vapor pressure data for 1,3-dinitrobenzene at 20°C (see Section 1.2.), the Henry's Law constant can be calculated to be 2.3×10^{-6} atm-m^3/mol. This value indicates a slow rate of volatilization from natural bodies of water (Lyman et al., 1982). Using a Henry's Law constant of 2.3×10^{-6} atm-m^3/mol, the U.S. EPA EXAMS computer simulation (Burns et al., 1981) predicts the following volatilization half-lives:

river	121 days
pond	225 days
oligotrophic lake	632 days

2.1.5.2. **ADSORPTION** -- Experimental data regarding adsorption of dinitrobenzenes to particulate organic matter and sediment in water were not located in the available literature as cited in the Appendix. Based on the K_{ow} and WS of 1,3-dinitrobenzene, the EXAMS computer simulation (Burns et al., 1981) predicts that >99% of the compound will remain in the water column and that <1% will be in the sediments in lakes.

2.1.5.3. **BIOACCUMULATION** -- Kawasaki (1980) used results of the MITI test to derive BCF values of <100 for the dinitrobenzenes in fish.

The BCF of an organic compound may be estimated from the K_{ow} by using the following equation (Veith et al., 1980): [Log BCF=0.76 Log K_{ow} - 0.23]. Using the Log K_{ow} values from Section 1.2., the BCFs for 1,2-, 1,3- and 1,4-dinitrobenzene are all calculated to be ~10. Estimation from WS may be done by using the following equation (Kenaga and Goring, 1978): [Log BCF=2.791-0.564 Log WS]. Using the WS values (in ppm) (see Section 1.2.) at 20-25°C, the BCFs for 1,2-, 1,3- and 1,4-dinitrobenzene are calculated to be ~8, 19 and 52, respectively. All of these BCF values suggest that the dinitrobenzenes are not expected to bioaccumulate significantly in aquatic organisms.

2.2. AIR

Pertinent data regarding the fate of the dinitrobenzenes in the atmosphere were not located in the available literature as cited in the Appendix. As noted in Section 2.1.3., the direct photolysis of 1,3-dinitrobenzene in aqueous solution may contribute to its disappearance from water. Lotz et al. (1983) exposed 1,2-dinitrobenzene to UV radiation of $\geq$290 nm. A 38% degradation of 1,2-dinitrobenzene was observed in 7 days. These results suggest that the photolysis of the dinitrobenzenes may occur significantly in air, but additional experimental data are required to ascertain its significance. Pertinent kinetic rate data regarding the reaction of dinitrobenzenes with hydroxyl radicals or ozone were not located in the available literature as cited in the Appendix.

2.3. SOIL

2.3.1. Microbial Degradation. Alexander and Lustigman (1966) examined the degradation of the three dinitrobenzenes by soil microflora from a Niagara silt loam soil. None of the dinitrobenzenes were completely degraded after 64 days of incubation, which suggested that they were

resistant to biodegradation. Other soil degradation studies were not located; additional data are required to determine the significance of the microbial degradation of the dinitrobenzenes in soil.

2.3.2. Chemical Degradation. Pertinent data regarding chemical degradation of dinitrobenzenes in soil were not located in the available literature as cited in the Appendix.

2.3.3. Photolysis. The photolysis data previously cited (see Section 2.1.3.2.2.) suggest that photolysis of the dinitrobenzenes from soil surfaces may not be significant; however, additional experimental data are required.

2.3.4. Adsorption/Leaching. Experimental adsorption/leaching studies regarding the dinitrobenzenes were not located in the available literature as cited in the Appendix. Prediction of the K_{oc} of an organic compound can be calculated from the WS by using the following equations:

$$\text{Log } K_{OC}=3.64-0.55 \text{ Log WS (in ppm)} \quad \text{(Kenaga and Goring, 1978)} \quad (2\text{-}1)$$

$$\text{Log } K_{OC}=0.44-0.54 \text{ Log WS (in mole fraction)} \quad \text{(Karickhoff et al., 1979)} \quad (2\text{-}2)$$

Using the WS values from Section 1.2., the K_{oc} values are calculated as follows:

	Equation 2-1	Equation 2-2
1,2-dinitrobenzene	65	257
1,3-dinitrobenzene	143	615
1,4-dinitrobenzene	392	1500

Predictions from the K_{ow} can be done from the following equation (Kenaga and Goring, 1978): $\text{Log } K_{oc}=0.544 \text{ Log } K_{ow}+1.377$. Using the Log

K_{ow} values from Section 1.2., the K_{oc} values for the dinitrobenzenes are calculated to range from 154-172. Prediction from the molecular topology and quantitative structure-activity relationship analysis method of Sabljic (1984) yields dinitrobenzene K_{oc} values >1000.

The K_{oc} estimations made above cover about two orders of magnitude, but generally indicate a moderate level of soil mobility. Experimental data are required to determine actual levels of leaching in the natural soil environment.

2.4. SUMMARY

Tenessee River water has been reported to rapidly degrade 1,3-dinitrobenzene by microbial decomposition (Mitchell and Dennis, 1982). The water has been exposed to upstream releases of the compound from TNT production operations suggesting that the microbes in the water have become adapted to 1,3-dinitrobenzene; other natural waters, not previously exposed to the compound, were unable to degrade it. Mixed culture studies using activated sludge or wastewater inocula have generally indicated that dinitrobenzenes are resistant to degradation.

Photolytic reactions of dinitrobenzenes in the environment may be significant but additional experimental data are required.

Hydrolysis and oxidation in water are not expected to be significant processes. 1,3-Dinitrobenzene is expected to volatilize slowly from water. Based on prediction equations, the dinitrobenzenes are not expected to significantly bioaccumulate in aquatic organisms.

Prediction equations suggest that dinitrobenzenes may have a moderate soil mobility. Experimental leaching studies are required to determine its significance.

3. EXPOSURE

3.1. WATER

DuPont reportedly generates 70-72 thousand pounds of 1,3-dinitrobenzene annually, which is consequently present in aqueous wastestreams resulting from the production of dinitrobenzene as well as nitrobenzene (TSCA-ITC, 1984).

The dinitrobenzenes are produced as by-products during the production of nitrobenzene (Dunlap, 1981), TNT explosive (Kitchens et al., 1979; Spanggord et al., 1980) and nitrobenzoic acid (TSCA-ITC, 1984). Aqueous wastestreams from these production processes are sources of exposure of the aquatic environment to dinitrobenzene. The 1,3-isomer of dinitrobenzene is the predominant by-product formed during these processes; ~93% of the dinitrobenzene by-products formed in TNT production is the 1,3-isomer (Kitchens et al., 1979). A concentration of 4 ppm 1,3-dinitrobenzene was detected in one aqueous wastestream from TNT production (Mitchell and Dennis, 1982). Kitchens et al. (1979) estimated that at full TNT production capacity, downstream river concentrations of 1,3-dinitrobenzene may range from 0.006-0.16 ppb.

In addition to release from industrial operations, 1,3-dinitrobenzene may be exposed to the environment by photochemical alteration of 2,4-dinitrotoluene, which is a by-product released to the environment from the manufacture of munitions (Kitchens et al., 1979).

The U.S. EPA STORET Data Base has no observations for the dinitrobenzenes.

3.2. FOOD

Pertinent data regarding dinitrobenzene exposure from food were not located in the available literature as cited in the Appendix.

3.3. INHALATION

DuPont reported that ~60 pounds of 1,3-dinitrobenzene are released into the atmosphere annually as a result of processing of the compound (TSCA-ITC, 1984). Atmospheric monitoring data were not located in the available literature as cited in the Appendix.

3.4. DERMAL

Pertinent data regarding dermal exposure to dinitrobenzenes were not located in the available literature as cited in the Appendix.

4. PHARMACOKINETICS

4.1. ABSORPTION

Parke (1961) treated rabbits by gavage with randomly-labeled 1,3-dinitro[^{14}C]benzene in arachis oil in single doses of 50-100 mg/kg. Within 2 days, 65-93% of the administered radioactivity was detected in the urine, and $\leq$5% was found in the feces, indicating that at least 65-93% of the dose was absorbed by the gastrointestinal tract.

Absorption of 1,3-dinitrobenzene after inhalation by humans can be inferred from the occupational exposure studies of Okubo and Shigeta (1982) and Bresson et al. (1965) (Section 5.6.). Percutaneous absorption of 1,3-dinitrobenzene by humans can be inferred from the report of occupational exposure by Ishihara et al. (1976) in which a worker developed methemoglobinemia after immersing her hands in a 0.5% w/w solution of 1,3-dinitrobenzene (Section 5.6.).

Pertinent data regarding the absorption of 1,2-dinitrobenzene or 1,4-dinitrobenzene were not located in the available literature as cited in the Appendix.

4.2. DISTRIBUTION

Pertinent data regarding the distribution of 1,2-dinitrobenzene, 1,3-dinitrobenzene or 1,4-dinitrobenzene were not located in the available literature as cited in the Appendix.

4.3. METABOLISM

In the previously described study (see Section 4.1.), Parke (1961) analyzed the urine collected for 2 days following gavage dosing of rabbits with 1,3-dinitro[^{14}C]benzene at 50-100 mg/kg. The results are given in Table 4-1. Based on the data of Parke (1961), Kitchens et al. (1979) developed the diagram of metabolic pathways presented in Figure 4-1. Parke

TABLE 4-1

Metabolites of 1,3-Dinitro[^{14}C]benzene Recovered from the Urine and Feces of Rabbits 2 Days After Oral Administration Expressed as Percentage of Administered Radioactivity[a]

Compound	Dose (mg/kg)							
	Average	50	50	60	60	70	70	100[b]
1,3-Dinitrobenzene	0.7	0.7	0.4	ND	2.4	ND	0.3	0.1
1,3-Nitrophenyl-hydroxylamine	0.8	0.75	1.1	0.7	ND	ND	ND	ND
1,3-Nitrosonitrobenzene	0.25	0.25	0.1	0.2	0.5	ND	ND	ND
3,3'-Dinitroazoxybenzene	0.3	ND	0.3	0.2	0.3	ND	ND	ND
1,3-Nitroaniline (total)	35	ND	ND	ND	18	10	35	28
1,3-Phenylenediamine (total)		ND	ND	ND	25	23	<0.2	<0.2
2,4-Dinitrophenol (total)	0.1	ND	ND	ND	ND	0.1	0.1	0.1
2-Amino-4-nitrophenol (total)	14	ND	ND	ND	16	12	15	12.5

TABLE 4-1 (cont.)

Compound	Dose (mg/kg)							
	Average	50	50	60	60	70	70	100[b]
4-Amino-2-nitrophenol (total)	2.0 (0.8)[c]	2.4 (1.0)[c]	1.9 (0.6)[c]	ND	2.6	2.1	1.6	1.4
2,4-Dinitrophenol (total)	31	ND	ND	ND	28	38	37	19
Total metabolites	83	ND	ND	ND	93	85	89	61
Total radioactivity in urine	81	75	89	71	93	82	89	65
Total radioactivity in feces	ND	ND	ND	5.2	ND	1.0	0.3	ND

[a]Source: Parke, 1961

[b]Animal died on the third day.

[c]Estimated after enzymatic hydrolysis

ND = None detected

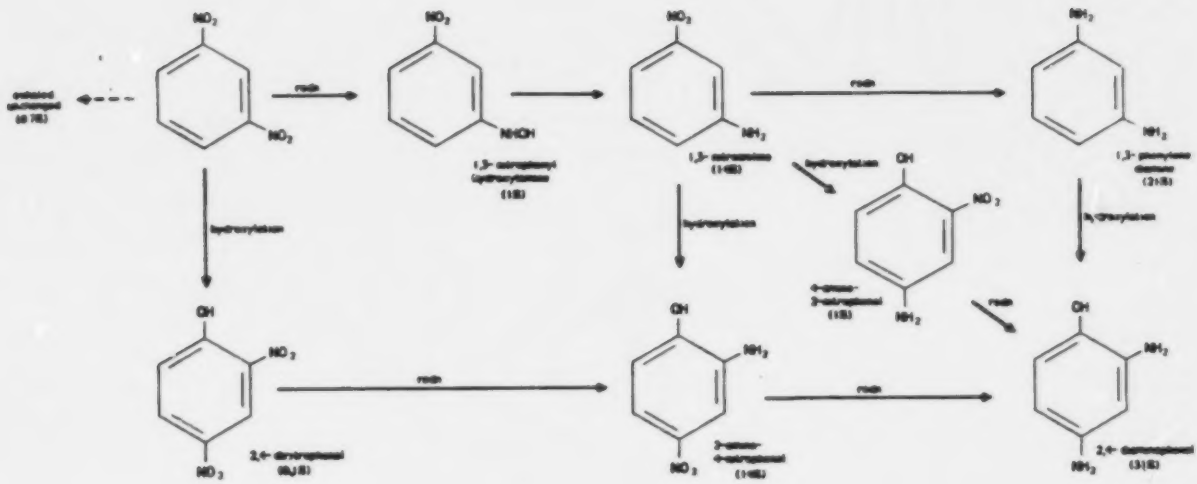


FIGURE 4-1

Metabolic Pathway of 1,3-Dinitrobenzene in Rabbits

Source: Kitchens et al., 1979

(1961) also treated rabbits with unlabeled 1,3-dinitrobenzene and found 30% of the dose was present as glucuronide conjugates and 6% as ethereal sulfate conjugates in the urine.

Pertinent data regarding the metabolism of 1,2-dinitrobenzene or 1,4-dinitrobenzene were not located in the available literature as cited in the Appendix.

4.4. EXCRETION

As discussed previously in Section 4.1. rabbits were treated by gavage with a single dose of 50-100 mg/kg of randomly labeled 1,3-dinitro[^{14}C]-benzene in arachis oil (Parke, 1961). Between 65 and 93% of the administered radioactivity was detected in the urine collected over 48 hours with only trace amounts of radioactivity found after 48 hours. Between 0.3 and 5.2% of the administered radioactivity was detected in the feces collected over 48 hours, however no radioactivity was detected in the feces of some rabbits.

Pertinent data regarding the excretion of 1,2-dinitrobenzene or 1,4-dinitrobenzene were not located in the available literature as cited in the Appendix.

5. EFFECTS

5.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of 1,2-dinitrobenzene, 1,3-dinitrobenzene or 1,4-dinitrobenzene were not located in the available literature as cited in the Appendix, and these compounds are not scheduled for testing by the National Toxicology Program (NTP, 1985).

5.2. MUTAGENICITY

As summarized in Table 5-1, 1,3-dinitrobenzene was mutagenic in Salmonella typhimurium strains TA98 and TA1538 with and without metabolic activation (Spanggord et al., 1982; Chiu et al., 1978; McGregor et al., 1980; Matsuda, 1981). 1,3-Dinitrobenzene was mutagenic in S. typhimurium strain TA100 in three of the four assays with or without metabolic activation (Spanggord et al., 1982; McGregor et al., 1980; Matsuda, 1981). Only Chiu et al. (1978) reported that 1,3-dinitrobenzene was not mutagenic in S. typhimurium strain TA100; evaluation was made only at one Log dose intervals with only 3 concentrations tested without metabolic activation.

In addition to the studies summarized in Table 5-1, Probst et al. (1981) reported that 1,3-dinitrobenzene and 1,4-dinitrobenzene did not increase unscheduled DNA synthesis in cultivated hepatocytes. 1,3-Dinitrobenzene, however, was positive for reverse mutations in S. typhimurium strains, TA1538, TA98 and D3052 and 1,4-dinitrobenzene was positive in strains TA100, TA1538 and TA98 with and without metabolic activation tested by plate incorporation. The 1,2-Dinitrobenzene was negative for reverse mutations in all bacterial strains and in the unscheduled DNA synthesis assay. An abstract of the work by Shah and Andrews (1979) indicated that 1,4-dinitrobenzene increased the frequency of mutations in S. typhimurium strains TA98, TA100 and TA1538 only in the presence of metabolic activators.

TABLE 5-1

Mutagenicity of 1,3-Dinitrobenzene

Assay	Indicator/ Organism	Concentration/ Dose	Application Method	Metabolic Activation	Response	Comments	Reference
Reverse mutation	Salmonella typhimurium	0.0-600.0 µg/plate	plate incorporation			The formation of Janovsky Type complexes may be required for the reduction of the nitro group.	Spanggord et al., 1982
	TA98			+/-S9	+/+		
	TA100			+/-S9	+/+		
	TA1535			+/-S9	-/-		
	TA1537			+/-S9	-/-		
	TA1538			+/-S9	+/+		
	TA100NR3			+/-S9	-/-		
Reverse mutation	S. typhimurium TA98	0.1, 1.0 and 10 µmole/plate	plate incorporation	-S9	+	Bacterial reduction of one or both nitro groups may have produced a compound that was responsible for the mutagenicity of 1,3-dinitrobenzene in strain TA98.	Chiu et al., 1978
	TA100			-S9	-		
Reverse mutation	S. typhimurium TA1538	50.0 and 100.0 µg/plate	plate incorporation	+/-S9	+/+	NC	Garner and Nutman, 1977
Reverse mutation	S. typhimurium TA98	1-1,000 µg/plate	plate incorporation	+/-S9	+/+	NC	McGregor et al., 1980
	TA100			+/-S9	+/+		
	TA1535			+/-S9	-/-		
	TA1537			+/-S9	+/+		
	TA1538			+/-S9	+/+		
Reverse mutation	S. typhimurium TA98	NR	NR	+/-S9	+/+	NC	Matsuda 1981
	TA100			+/-S9	+/+		

NC = No comment; NR = not reported; NR3 = Nitroreductase-deficient strain of TA100

0670H

5.3. TERATOGENICITY

Pertinent data regarding the teratogenicity of 1,2-dinitrobenzene, 1,3-dinitrobenzene and 1,4-dinitrobenzene were not located in the available literature as cited in the Appendix.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding the other reproductive effects of 1,2-dinitrobenzene, 1,3-dinitrobenzene and 1,4-dinitrobenzene were not located in the available literature as cited in the Appendix.

5.5. CHRONIC AND SUBCHRONIC TOXICITY

Groups of 20 male and 20 female Carworth Farms rats were exposed to 0, 3, 8 or 20 ppm of 1,3-dinitrobenzene in their drinking water for 16 weeks (Christian et al., 1976; Cody et al., 1981). Peripheral blood samples were taken from 6 males/group after 5, 10 and 14 weeks of treatment. Hematocrit and hemoglobin levels were determined and white blood cell counts and differentials were performed on five male rats 5, 10 and 14 weeks after the start of the experiment. A record of body weight and water and food consumption was kept throughout the experiment and used to determine the exposure level of 1,3-dinitrobenzene, as summarized in Table 5-2. After termination, body and organ weights were recorded and histopathological examination of major organs was performed. No effects were observed at 3 ppm. Significantly increased splenic weight was observed at 8 ppm. Female rats at 20 ppm had significantly decreased body weight gain compared with controls. Although food consumption was also decreased, Cody et al. (1981) concluded that 1,3-dinitrobenzene consumption was at least partially responsible for the decreased body weight observed in the high dose females. Decreased body weight or food consumption was not observed in the male rats or in females at lower doses. There was a significant

TABLE 5-2

The Average Daily Intake of 1,3-Dinitrobenzene in Rats*

Exposure Level in Drinking Water (ppm)	Dose for Females (mg/kg/day)	Dose for Males (mg/kg/day)
3	0.48±0.07	0.40±0.09
8	1.32±0.19	1.13±0.244
20	3.10±0.425	2.64±0.637

*Source: Cody et al., 1981

decrease (60%) in testes weight accompanied by "moderate" atrophy in the male rats at 20 ppm.

A significant increase in the weight of the ovaries in female rats exposed to 3 ppm of 1,3-dinitrobenzene was observed, but no significant effect on ovarian weight was observed in the rats exposed to higher levels of 1,3-dinitrobenzene. Several hematological parameters were affected during this study. The hemoglobin count was significantly decreased in male rats at 20 ppm at 5 and 10, but not 14 weeks after the start of treatment. Although the hematocrit was decreased in the 20 ppm males 5 weeks after the start of 1,3-dinitrobenzene treatment, the decrease was not statistically significantly and no significant differences were observed between treated and control rats after 10 and 14 weeks of treatment. The leukocyte count was significantly decreased in male rats at 3 and 8 ppm 10 weeks after the start of treatment; however, because a significant effect on leukocyte count was not observed at any other dose or time interval, the leukopenia observed 10 weeks after the start of treatment was not considered treatment-related. Blood parameters were not evaluated in female rats. Both male and female rats treated with 20 ppm of 1,3-dinitrobenzene had significantly increased splenic weights accompanied by "pronounced" hemosiderin deposits (Christian et al., 1976; Cody et al., 1981).

The average length of a sleep period resulting from a standard dose of sodium hexobarbital was significantly longer in males at 8 ppm; however, because no significant effect on sleep period was observed at 3 or 20 ppm in males or in the females at any dose tested, the effect of 1,3-dinitrobenzene on the length of the drug-induced sleeping period was not considered treatment-related (Cody et al., 1981; Christian et al., 1976). Furthermore, hexabarbital sleeping time was significantly decreased in females at ≥50 ppm and at ≥100 ppm in males in a 7 week study (Cody et al., 1981).

In another experiment, groups of 10 male rats were exposed to 0, 3 or 8 ppm of 1,3-dinitrobenzene in the drinking water for 90 days (Cody et al., 1981). Significantly increased wheel running activity was observed in the rats exposed to both levels of 1,3-dinitrobenzene. Although activity platform counts were also increased in rats exposed to both levels of 1,3-dinitrobenzene, the increase was not statistically significant (Cody et al., 1981).

Cody et al. (1981) also treated groups of 6 male and 6 female Carworth Farm rats with 1,3-dinitrobenzene in the drinking water at concentrations of 50, 100 or 200 ppm for 8 weeks. Controls consisted of 12 males and 12 females maintained on tap water. Mortality was high at 200 ppm. There was a dose-related decrease in body weight gain as well as in reduced food and water consumption in males and females. Significantly reduced hemoglobin concentrations were observed in males and females at ≥50 ppm compared with controls. Hexobarbitol sleeping time was significantly decreased in females at all exposure levels and in males at 100 ppm. Increased relative spleen weight in males and females at ≥50 ppm and decreased relative testes weight at ≥50 ppm were observed. Histologic examination revealed a dose-related increased severity of deposition of a brown-yellow pigment in Kupffer cells of the liver at ≥50 ppm. Decreased spermatogenesis as evidenced by reduced activity of spermatogenic epithelium activity and decreased number of germinal cells was observed at 50 and 100 ppm. At 200 ppm, seminiferous tubules were collapsed and lacked germinal and Sertoli cells in males, and there was atrophy, fibrosis and hemosiderosis in the spleen of both sexes.

5.6. OTHER RELEVANT INFORMATION

The acute toxicity of 1,3 dinitrobenzene is summarized in Table 5-3. Application of 1,3-dinitrobenzene to the skin of cats resulted in methemoglobinemia within 24 hours (White and Hay, 1901). As reviewed by TSCA-ITC (1984), Ishihara and Ikeda (1979) reported that the composition of the vehicle had an effect on the percutaneous absorption of 1,3-dinitrobenzene in rats. Guinea pigs developed methemoglobinemia after treatment with a 0.5% solution of 1,3-dinitrobenzene in 87.5% ethylene glycol, 5% water and 7% ammonium adipate, but did not develop methemoglobinemia after treatment under identical conditions with a 0.5% solution of 1,3-dinitrobenzene in 62.5% ethylene glycol, 30% diethylene glycol and 7% ammonium sebacate. Rats treated orally at 83 mg/kg of 1,3-dinitrobenzene developed reduced ambulatory motion, ataxia, weakness, dyspnea, rapid heartbeat, cyanosis, respiratory failure and coma (Cody et al., 1981).

As summarized by TSCA-ITC (1984), cyanosis was a characteristic effect observed in humans exposed to 1,3-dinitrobenzene (Okubo and Shigeta, 1982; Bresson et al., 1965; Ishihara et al., 1976). The onset of cyanosis ranged from 1 day to several weeks after initial exposure to 1,3-dinitrobenzene. Other effects reported in humans included methemoglobinemia, Heinz body formation and anemia. Although two reports (Okubo and Shigeta, 1982; Bresson et al., 1965) indicated that no liver damage occurred after inhalation of 1,3-dinitrobenzene. Levels of inhalation exposure were not specified. A third report (Ishihara et al., 1976) indicated that a woman exposed percutaneously to 1,3-dinitrobenzene had an icteric face and sclera, and a palpable liver. The woman, exposed dermally, had handled electronic parts that had been immersed in a solution containing 0.5% w/w 1.3-dinitrobenzene for 0.5 to 0.75 hours/day for 3 days. The other components of the solution were ethylene glycol (87.5%), water (5%) and ammonium adipate (7%).

TABLE 5-3

Acute Toxicity of 1,3-Dinitrobenzene

Species	Sex	Route	LD_{50} (mg/kg)	Reference
Rat	M/F	oral	83	Cody et al., 1981
Rat	M	oral	91	Cody et al., 1981
Rat	F	oral	81	Cody et al., 1981
Rat	NR	intraperitoneal	25.2	Pankow et al., 1975
Dog	NR	subcutaneous	10.0	Kiese, 1949

NR = Not reported

Although nitrobenzene did not induce methemoglobinemia in antibiotically pretreated rats, pretreatment of rats with antibiotics had no effect on the induction of methemoglobinemia by 1,2-, 1,3- or 1,4-dinitrobenzene, indicating that induction of methemoglobinemia is independent of the gut microflora (Facchini and Griffiths, 1981).

Rats treated with intraperitoneal injections of 0.015 mmol/kg of 1,4-dinitrobenzene, 0.15 mmol/kg of 1,3-dinitrobenzene or 0.45 mmol/kg of 1,2-dinitrobenzene excreted increased amounts of urinary catecholamine and had elevated blood sugar levels (Pankow and Ponsold, 1978).

6. AQUATIC TOXICITY

6.1. ACUTE EFFECTS

In a battery of static acute toxicity tests of 1,3-dinitrobenzene, van der Schalie (1983) reported 96-hour LC_{50} values of 1.44, 1.70, 8.13 and 16.8 mg/ℓ in bluegill sunfish, Lepomis macrochirus, rainbow trout, Salmo gairdneri, channel catfish, Ictalurus punctatus, and fathead minnows, Pimephales promelas. Static 96-hour toxicity tests of 1,3-dinitrobenzene were conducted using fathead minnows, P. promelas, in soft water (40-48 mg $CaCO_3$/ℓ) at 22°C. A 96-hour LC_{50} value of 12.7 mg 1,3-dinitrobenzene/ℓ was calculated from concentration-mortality data (Curtis and Ward, 1981). Liu et al. (1983a,b) reported a 96-hour LC_{50} of 7.0 mg 1,3-dinitrobenzene/ℓ in P. promelas. Holcombe et al. (1984) reported flow through 24-, 48-, 72- and 96-hour LC_{50} values of 0.687, 0.641, 0.609 and 0.603 mg/ℓ, respectively, for 1,4-dinitrobenzene. Bailey and Spanggord (1983) reported static 96-hour LC_{50} values of 0.6, 7.0 and 1.7 mg/ℓ, for 1,2-, 1,3- and 1,4-dinitrobenzene, respectively, in fathead minnows, P. promelas. LeClerc (1960) reported the lethality threshold of dinitrobenzene (specific isomer or mixture not specified) in redbelly dace minnows, Chrosomus sp., was 10-12 mg/ℓ in distilled water following 6-hour exposures at 23°C. In hard water, the minimum lethal dose was 8-10 mg/ℓ for 6-hour exposures. In the golden orfe, Leuciscus idus melanotus, Juhnke and Luedemann (1978) determined 48-hour LC_0, LC_{50} amd LC_{100} values of 3, 10 and 20 mg/ℓ, respectively.

Liu et al. (1983a,b) reported acute toxic effects in the freshwater crustacean, Daphnia magna, after exposure at levels of ~50 mg 1,3-dinitrobenzene/ℓ. A 48-hour LC_{50} at 49.6 mg/ℓ was reported. van der Schalie (1983) reported a 48-hour EC_{50} of 27.4 mg 1,3-dinitrobenzene/ℓ for loss of equilibrium in D. magna.

In freshwater snails, Lymnae sp., exposed to 10 mg technical 1,3-dinitrobenzene/ℓ, mortality was <100% (Batte et al., 1951). Data regarding the test duration and other mortality data were not available.

6.2. CHRONIC EFFECTS

Rainbow trout were exposed at 0.16, 0.42, 0.76, 1.42 and 3.00 mg 1,3-dinitrobenzene/ℓ for up to 30 days (van der Schalie, 1983). LT_{20} values were 4, 8, 10, and 20 days after exposure at 3.00, 1.42, 0.76 and 0.42 mg/ℓ, respectively. No mortality occurred among fish exposed at 0.16 mg/ℓ, or in water or solvent control groups within 30 days. The estimated 30-day LC_{50} was 0.37 mg 1,3-dinitrobenzene/ℓ.

Hermens and Leeuwangh (1982) reported that the 14-day LC_{50} value for 1,3-dinitrobenzene was 9.4 mg/ℓ for the guppy, Poecilia reticulata.

van der Schalie (1983) reported two embryo-larval toxicity tests of 1,3-dinitrobenzene in rainbow trout. Tests were conducted for 68-69 days, allowing 60 days of exposure after hatching. Test concentrations ranged from 0.08-0.97 mg 1,3-dinitrobenzene/ℓ. Effect on fry survival was the most sensitive parameter measured, with marginally significant ($p<0.05$) inhibition at levels $\geq$0.50 mg 1,3-dinitrobenzene/ℓ. A level of 0.44 mg/ℓ was the highest NOEC. It was noted by van der Schalie (1983) that the 30-day LC_{50} value for adult rainbow trout (0.37 mg/ℓ) was actually lower than the NOEC for eggs and fry.

6.3. PLANT EFFECTS

During assays conducted to identify possible algicides, Fitzgerald et al. (1952) reported that 5 mg 1,3-dinitrobenzene/ℓ caused 50% mortality in cultures of the blue-green algae, Microcystic aeruginosa. Bringmann and Kuehn (1978) reported a toxicity threshold level of 0.17 mg 1,3-dinitrobenzene/ℓ for inhibited cell multiplication in M. aeruginosa. The toxicity

threshold for cell multiplication inhibition in the green algae, Scenedesmus quadricauda, following exposure for 7 days, was 0.7 mg 1,3-dinitrobenzene/ℓ (Bringmann and Kuehn, 1978, 1980). Growth inhibition of Selenastrum capricornutum occurred at concentrations >0.26 mg 1,3-dinitrobenzene/ℓ during 5-day exposures. Levels ≥85.6 mg/ℓ were determined to be algicidal, while levels as low as 0.97 mg/ℓ appeared to be algistatic (van der Schalie, 1983).

6.4. RESIDUE

Pertinent data regarding the residue of dinitrobenzenes in aquatic organisms or the aquatic environment were not located in the available literature as cited in the Appendix.

6.5. OTHER RELEVANT INFORMATION

Bringmann and Kuehn (1980) reported toxicity threshold levels for inhibited multiplication at 0.76 mg/ℓ in the protozoan, Entosiphon sulcatum, and 14.0 mg/ℓ in the bacteria, Pseudomonas putida.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

The American Conference of Governmental Industrial Hygienists (ACGIH, 1984) adopted a TWA-TLV of 1 mg/m³ (0.15 ppm) of dinitrobenzene (all isomers) and a 15-minute STEL of 3 mg/m³ (0.5 ppm), based on analogy to the toxicity of aniline (ACGIH, 1980). The Occupational Safety and Health Administration (OSHA) standard for dinitrobenzene (all isomers) exposure in the workplace requires that the concentration not exceed 1 mg/m³ on an 8-hour TWA basis (CFR, 1981). Both the OSHA standard and the ACGIH criterion are followed by the skin notation emphasizing the importance of the cutaneous route, including mucous membranes and eyes, in the absorption of all three dinitrobenzene isomers.

7.2. AQUATIC

Guidelines and standards for the protection of aquatic organisms from the toxic effects of dinitrobenzenes were not located in the available literature as cited in the Appendix.

8. RISK ASSESSMENT

The carcinogenicity of any of the dinitrobenzene isomers has not been adequately studied. The dinitrobenzene isomers are not scheduled for carcinogenicity testing by NTP (1985). 1,3-Dinitrobenzene was positive for reverse mutation in Salmonella typhimurium strains TA98, TA100, TA1538 and possibly TA1537 with and without metabolic activation by the S9 microsomal fraction (see Table 5-1). Positive results for reverse mutation were found for 1,2-dinitrobenzene in S. typhimurium strains TA1538, TA98, TA100 and D3052 and for 1,4-dinitrobenzene in strains TA98, TA100 and TA1538 (Probst et al., 1981). None of the dinitrobenzene isomers increased unscheduled DNA synthesis in cultivated hepatocytes ; Probst et al., 1981). Pertinent data regarding either the teratogenicity or other reproductive effects of 1,2-dinitrobenzene, 1,3-dinitrobenzene or 1,4-dinitrobenzene were not located in the available literature as cited in the Appendix.

In an 8-week study, rats were given drinking water containing 50, 100 and 200 ppm 1,3-dinitrobenzene (Cody et al., 1981). Effects included markedly reduced body weight gain associated with reduced food and water consumption, significantly reduced hemoglobin concentrations, decreased hexobarbitol sleeping time, increased spleen weight, decreased testes weight, deposition of yellow-brown pigment in Kupffer cells and decreased spermatogenesis at $\geq$50 ppm. At 200 ppm, complete loss of spermatogenesis and collapse of seminiferous tubules in males and atrophy, fibrosis and hemosiderosis of the spleen in both sexes were observed.

The subchronic studies of Cody et al. (1981) and Christian et al. (1976), in which rats were exposed to 0, 3, 8 or 20 ppm of 1,3-dinitrobenzene in their drinking water for 13-16 weeks, can be used to derive an ADI of 0.0004 mg/kg/day or 0.028 mg/day for a 70 kg man. The endpoints

evaluated by Cody et al. (1981) and Christian et al. (1976) included body and organ weights, histopathology, blood hemoglobin, hematocrit and white blood cell values, and behavioral modification as measured by running wheel and platform activity. At 20 ppm, decreased body weight of females, testicular atrophy in males and increased spleen weights and hemosiderosis in both sexes were observed. Significantly increased splenic weight occurred in both male and female rats exposed to 8 ppm of 1,3-dinitrobenzene in the drinking water. At the next lower dose tested (3 ppm), the only observed effect was increased activity on the running wheel. Although increased running wheel activity may reflect an effect of 1,3-dinitrobenzene on the CNS, it does not appear to be an adverse effect. Therefore, exposure of rats to 3 ppm of 1,3-dinitrobenzene in the drinking water for 16 weeks is judged to be a NOAEL. Based on water consumption data, Cody et al. (1981) calculated that rats receiving 3 ppm consumed 1,3-dinitrobenzene at a dosage of 0.40 mg/kg/day; at the LOAEL of 8 ppm, the dosage was 1.13 mg/kg/day. To derive the chronic human ADI of 0.028 mg/day, the animal NOAEL of 0.40 mg/kg/day was multiplied by the assumed weight of a man (70 kg) and divided by an uncertainty factor of 100 to account for interspecies extrapolation and the range of human sensitivity to xenobiotics and the use of a subchronic study to derive a chronic human ADI.

The chronic ADI for 1,2-dinitrobenzene and 1,4-dinitrobenzene is also 0.028 mg/day, based on analogy to 1,3-dinitrobenzene.

9. REPORTABLE QUANTITIES

9.1. REPORTABLE QUANTITY (RQ) RANKING BASED ON CHRONIC TOXICITY

As discussed in Section 5.5., exposure of rats to drinking water containing 1,3-dinitrobenzene resulted in several significant effects (Cody et al., 1981; Christian et al., 1976).

Exposure of male rats to 3 ppm of 1,3-dinitrobenzene in the drinking water for 90 days resulted in significantly increased running wheel activity. Exposure of male and female rats to 8 ppm of 1,3-dinitrobenzene in the drinking water for 16 weeks resulted in significantly increased splenic weight. Exposure of male rats to 20 ppm of 1,3-dinitrobenzene in the drinking water for 16 weeks resulted in significantly decreased testicular weight accompanied by testicular atrophy. The actual dose, in mg/kg/day of 1,3-dinitrobenzene, that the rats received was calculated by Cody et al. (1981), summarized in Table 5-2 and used to derive the chronic human MEDs reported in Table 9-1. The source of the discrepancy between the MED reported in Table 9-1 and the MED's reported by U.S. EPA (1983) is not apparent and soes not affect either the CS on the RQ.

To derive the chronic human MED, the animal dose in mg/kg/day was multiplied by the cube root of the ratio of the animal body weight-to-human body weight and by the human body weight, and divided by 10 to account for using a subchronic study to derive a chronic MED. The average body weight of the rats was assumed to be 0.35 kg and of humans to be 70 kg. The RV_d associated with the MED for each effect is listed in Table 9-1.

It is appropriate to derive the RQ for 1,3 dinitrobenzene exposoure from the Human MED giving the highest CS. The CS's of 6, 21.2 and 33.6 were obtained by multiplying the RVd by the RVe and correspond to RQ's of 1000, 100, and 100 respectively (see Table 9-1). Although the RQ associated with

TABLE 9-1

Composite Scores for the Oral Toxicity of 1,3-Dinitrobenzene in Rats[a]

Exposure/ Dose	Human MED[b] (mg/day)	RV_d	Effects	RV_e	CS	RQ
3 ppm, 0.4 mg/kg/day for 90 days	0.48	6.0	Increased physical activity as measured by a running wheel	1	6	1000
8 ppm 1.13 mg/kg/ day for 16 weeks	1.35	5.3	Significantly increased splenic weight in males	4	21.2	100
20 ppm 2.64 mg/kg/ day for 16 weeks	3.16	4.8	Significantly decreased testicular weight and "moderate" atrophy	7	33.6	100

[a]Source: Cody et al., 1981

[b]The dose was divided by an uncertainty factor of 10 to approximate chronic exposure.

splenic effects and the RQ associated with testicular effects are both 100, the highest CS (33.6) is obtained for testicular effects. Therefore, an RQ of 100 based on decreased testicular weight and testicular atrophy in rats (Cody et al., 1981) is recommended for 1,3 dinitrobenzene (Table 9-2).

Both the ACGIH (1980, 1983) and the U.S. EPA (1985) derived standards for 1,2-dinitrobenzene and 1,4-dinitrobenzene by analogy to 1,3-dinitrobenzene. Following similar reasoning, the RQ for 1,2-dinitrobenzene and for 1,4-dinitrobenzene would be 100 based on the toxicological information available for 1,3-dinitrobenzene.

9.2. WEIGHT OF EVIDENCE AND POTENCY FACTOR ($F=1/ED_{10}$) FOR CARCINOGENICITY

Because of the lack of pertinent data regarding the carcinogenicity of the dinitrobenzene isomers, a potency factor ($1/ED_{10}$) cannot be derived. The dinitrobenzenes cannot be classified as to their carcinogenic potential for humans and, therefore, can be placed in IARC group 3 on U.S. EPA Group D.

TABLE 9-2

1-3-Dinitrobenzene

Minimum Effective Dose (MED) and Reportable Quantity (RQ)

Route:	oral
Dose*	3.16 mg/kg
Effect	significantly decreased testicular weight accompanied by testicular atrophy and possible loss of function
Reference:	Cody et al., 1981
RV_d	4.8
RV_e	7
Composite Score:	33.6
RQ:	100

*Equivalent human dose

10. REFERENCES

ACGIH (American Conference of Governmental Industrial Hygienists). 1980. Documentation of the Threshold Limit Values, 4th edition. (Includes Supplemental Documentation, 1981, 1982, 1983.) Cincinnati, OH. p. 151-152.

ACGIH (American Conference of Governmental Industrial Hygienists). 1984. Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1984-1985. Cincinnati, OH. p. 17.

Alexander, M. and B.K. Lustigman. 1966. Effect of chemical structure on microbial degradation of substituted benzenes. J. Agric. Food Chem. 14: 410-413.

Bailey, H.C. and R.J. Spanggord. 1983. The relationship between the toxicity and structure of nitroaromatic chemicals. Aquatic Toxicology and Hazard Assessment: Sixth Symposium ASTM STP 802 W.E. Bishop, R.D. Cardwell and B.B. Heidolph, Ed. American Society for Testing and Materials, Philadelphia, PA. p. 98-107.

Batte, E.G., L.E. Swanson and J.B. Murphy. 1951. New molluscicides for the control of freshwater snails. Am. J. Vet. Res. 12(43): 158-160. (Cited in Battelle's Columbus Laboratories, 1971)

Battelle's Columbus Laboratories. 1971. Water Quality Criteria Data Book. Vol. 3. Effects of Chemicals on Aquatic Life. Selected Data from the Literature through 1968. Prepared for the U.S. EPA under Contract No. 68-01-0007. Washington, DC.

Bresson, J.R., R. Gerard, J. Leder and F. Tolot. 1965. Acute poisoning in workers exposed to derivatives of nitrobenzene. Arch. Mal. Prof. Med. Trav. Sci. Soc. 27: 727-729. (Fre.) (Cited in TSCA-ITC, 1984)

Bringmann, G. and R. Kuehn. 1971. Biological decomposition of nitrotoluenes and nitrobenzenes by Azotobacter agilis. Gesundh. Ing. 92: 273-276.

Bringmann, G. and R. Kuehn. 1978. Grenzwerte der Schadwirkung wassergefihrdender Stoffe gegen Blaualgen (Microcystis aeruginosa) And Grunalgen (Scenedesmus quadricauda) in Zellvermehrangshemmtest. Vom. Wasser. 50: 45-60. (Ger.)

Bringmann, G. and R. Kuehn. 1980. Comparison of the toxicity thresholds of water pollutants to bacteria, algae, and protozoa in the cell multiplication inhibition test. Water Research. 14: 231-241.

Burns, L.H., D.M. Cline and R.R. Lassiter. 1981. Exposure Analysis Modeling Systems (EXAMS). Prepared by ERL, ORD, U.S. EPA, Athens, GA.

Cartwright, N.J. and R.B. Cain. 1959. Bacterial degradation of the nitrobenzoic acid. II. Reduction of the nitro group. Biochem. J. 73: 305-314. (Cited in Spanggord et al., 1980)

CFR (Code of Federal Regulations). 1981. OSHA Safety and Health Standards. (29 CFR 1910.1000)

Chambers, C.W., H.H. Tabak and P.W. Kabler. 1963. Degradation of aromatic compounds by phenol-adapted bacteria. J. Water Pollut. Control Fed. 35: 1517-1528.

Chiu, C.W., L.H. Lee, C.Y. Wang and G.T. Bryan. 1978. Mutagenicity of some commercially available nitro compounds for Salmonella typhimurium. Mutat. Res. 58: 11-22.

Christian, R.T., C.S. Clark, T.E. Cody, et al. 1976. The development of a test for the potability of water treated by a direct reuse system. Report No. 8. Department of Environmental Health, College of Medicine, University of Cincinnati. Contract No. DADA 17-73-C-3013. NTIS ADA 0225 143. (Cited in Wentsel et al., 1979)

Cody, T.E., S. Witherup, L. Hastings, K. Stemmer and R.T. Christian. 1981. 1,3-Dinitrobenzene: Toxic effect in vivo and in vitro. J. Toxicol. Environ. Health. 7(5): 829-847.

Curtis, M.W. and C.H. Ward. 1981. Aquatic toxicity of forty industrial chemicals: Testing in support of hazardous substance spill prevention regulation. J. Hydrology. 51: 359-367.

Dunlap, K.L. 1981. Nitrobenzenes and nitrotoluenes. In: Kirk-Othmer Encyclopedia of Chemical Technology, 3rd ed., Vol. 15, M. Grayson and D. Eckroth, Ed. John Wiley and Sons, Inc., New York. p. 920.

Durkin, P.R., J. Colman and K. Blackburn. 1982. Document Modification and Review of Health Effects Assessments. Prepared by Syracuse Research Corporation, Syracuse, NY, under Contract 68-03-3112. ECAO, U.S. EPA, Cincinnati, OH.

Facchini, V. and L.A. Griffiths. 1981. The involvement of the gastrointestinal microflora in nitro-compound-induced methemoglobinemia in rats and its relationship to nitro-group reduction. Biochem. Pharmacol. 30(9): 931-935.

Fitzgerald, G.P., G.C. Gerloff and F. Skoog. 1952. Studies on chemicals with selective toxicity to blue-green algae. Sewage Ind. Wastes. 24: 888-896.

Garner, R. and C.A. Nutmann. 1977. Testing of some azo dyes and their reduction products for mutagenicity using Salmonella typhimurium TA1538. Mutat. Res. 44: 9-19.

Hallas, L.E. and H. Alexander. 1983. Microbial transformation of nitroaromatic compounds in sewage effluent. Appl. Environ. Microbial. 45(4): 1234-1241.

Hansch, C. and A.J. Leo. 1981. Medchem Project. Issue No. 19. Pomana College, Claremount, CA.

Hermens, J. and P. Leeuwangh. 1982. Joint toxicity of mixtures of 8 and 24 chemicals to the guppy (Poecilia reticulata). Ecotoxicol. Environ. Safety. 6(3): 302-310.

Holcombe, G.W., G.L. Phipps, M.L. Knuth and T. Falhaber. 1984. The acute toxicity of selected substituted phenols, benzenes and benzoic acid esters to fathead minnows (Pimephales promelas). Environ. Pollut. Ser. A. 35(4): 367-381.

Ishihara, N. and M. Ikeda. 1979. Effects of solvents and solutes on the percutaneous absorption of m-dinitrobenzene. Int. Arch. Occup. Environ. Health. 44: 91-98. (Cited in TSCA-ITC, 1984)

Ishihara, N., A. Kanaya and M. Ikeda. 1976. m-Dinitrobenzene intoxication due to skin absorption. Int. Arch. Occup. Environ. Health. 36(3): 161-168. (CA 85:57687p)

Juhnke, I. and D. Luedemann. 1978. Results of the investigation of 200 chemical compounds for acute fish toxicity with the golden orfe test. Z. f. Wasser-und Abwasser-Forschung. 11(5): 161-164.

Karickhoff, S.W., D.S. Brown and T.A. Scott. 1979. Sorption of hydrophobic pollutants on natural sediments. Water Res. 13: 241-248.

Kawasaki, M. 1980. Experiences with the test scheme under the chemical control law of Japan: An approach to structure-activity correlations. Ecotoxic. Environ. Safety. 4: 444-454.

Kenaga, E.E. and C.A.I. Goring. 1978. Relationship Between Water Solubility, Soil Sorption, Octanol-Water Partitioning and Bioconcentration of Chemicals in Biota. Third Aquatic Toxicology Symposium, American Society for Testing and Materials, New Orleans, LA. October 17-18.

Kiese, M. 1949. Pharmakologische Untersuchungen über m-Dinitrobenzene. I. Akute Vergiftung mit m-Dinitrobenzol. Naunyn-Schmiedebengs Arch. Exp. Pathol. Pharmakol. 206: 361-383.

Kitano, M. 1978. Biodegradation and bioaccumulation test on chemical substances. OECD Tokyo Meeting, Reference Book TSU-No. 3.

Kitchens, J.F., W.E. Harward, III, D.M. Lauter, R.S. Wentsel and R.S. Valentine. 1979. Preliminary problem definition study of 48 munitions related chemicals. Volume I. Explosives related chemicals. NTIS AD-A066407.

LeClerc, E. 1960. The self-purification of streams and the relationship between chemical and biological tests. In: Proc. 2nd Symp. Treatment of Waste Waters, Pergamon Press, London. p. 281. (Cited in McKee and Wolf, 1963)

Liu, D.H.W., R.J. Spanggord, H.C. Bailey, H.S. Javitz and D.C.L. Jones. 1983a. Toxicity of TNT Wastewaters to Aquatic Organisms. Vol. II. Acute Toxicity of Condensate Wastewater and 2,4-Dinitrotoluene. Draft Final Report. SRI International, Menlo Park, CA. DAMD17-75-C-5056. (Cited in van der Schalie, 1983)

Liu, D.H.W., H.C. Bailey and J.G. Pearson. 1983b. Toxicity of a Complex Munitions Wastewater to Aquatic Organisms. Aquatic Toxicology and Hazard Assessment: Sixth Symposium. ASTM STP 802, W.E. Bishop, R.D. Cardwell and B.B. Heidolph, Ed. American Society for Testing and Materials, Philadelphia, PA. p. 135-150.

Lotz, F., J. Kempny and R.S. Graelis de Kempny. 1983. Light-induced decomposition of absorbed chemicals. A simple device for comparative studies. Chemosphere. 12(6): 873-878.

Lyman, W.J., W.F. Reehl and D.H. Rosenblatt. 1982. Handbook of Chemical Property Estimation Methods. McGraw-Hill Book Co., New York. p. 7-4, 15-16.

Malaney, G.W. and R.E. McKinney. 1966. Oxidative abilities of benzene-acclimated activated sludge. Water Sewage Works. 113: 302-309.

Matsuda, A. 1981. Studies on the mutagenicity of nitroaromatic compounds in environment. Gifu Gaigaku Igakubu Kiyo. 29(2): 278-296 (CA 95:75104g)

McCormick, N.G., F.E. Feeherry and H.S. Levinson. 1976. Microbial transformation of 2,4,6-trinitrotoluene and other nitroaromatic compounds. Appl. Environ. Microbiol. 31(6): 949-958.

McGregor, D.B., C.G. Riach, R.M. Hastwell and J.C. Dacre. 1980. Genotoxic activity in microorganisms of tetryl, 1,3-dinitrobenzene and 1,3,5-trinitrobenzene. Environ. Mutagen. 2(4): 531-541. (CA 94:133412y)

McKee, J.E. and H.W. Wolf. 1963. Water Quality Criteria, 2nd ed. Prepared for the Resources Agency of California State Water Quality Control Board. Publ. No. 3-A.

Mitchell, W.R. and W.H. Dennis, Jr. 1982. Biodegradation of 1,3-dinitrobenzene. J. Environ. Sci. Health. A17: 837-853.

Naumova, R.P., N.N. Amerkhanova and T.O. Belousova. 1982. Reductive transformation of aromatic nitro compounds by bacteria. Mikrobiologiya. 51(5): 735-739. (CA 98:31121t)

NIOSH (National Institute for Occupational Safety and Health). 1979. 1978 Registry of Toxic Effects of Chemical Substances. U.S. OHEW, PHS, CDC, NIOSH, Cincinnati, OH. p. 180-181.

NTP (National Toxicology Program). 1985. Management Status Report. Dated 4/08/85.

Okubo, T. and S. Shigeta. 1982. Anemia cases after acute m-dinitrobenzene intoxication due to an occupational exposure. Ind. Health. 20(4): 297-304. (CA 98:77485t)

Pankow, D. and W. Ponsold. 1978. Urinary catecholamine excretion and blood sugar response during acute poisonings with dinitrobenzenes. Toxicology. 11(4): 377-384.

Pankow, D., W. Glatzel, K. Tietze and W. Ponsold. 1975. Motor nerve conduction velocity after carbon monoxide on m-dinitrobenzene poisonings. Arch. Toxicol. 34: 325-330

Parke, D. 1961. Studies in detoxification. V. The metabolism of m-dinitro[^{14}C]benzene in the rabbit. Biochem. J. 78: 262-271.

Pitter, P. 1976. Determination of biological degradability of organic substances. Water Res. 10: 231-235.

Probst, G.S., R.E. McMahon, L.E. Hill, C.Z. Thompson, J.K. Epp and S.B. Neal. 1981. Chemically-induced unscheduled DNA synthesis in primary rat hepatocyte cultures: A comparison with bacterial mutagenicity using 218 compounds. Environ. Mutagen. 3(1): 11-32.

Sabljic, A. 1984. Predictions of the nature and strength of soil sorption of organic pollutants by molecular topology. J. Agric. Food Chem. 32: 243-246.

Shah, M.J. and A.W. Andrews. 1979. Mutagenic evaluation of oxidation products of p-phenylene-diamine. A hair dye component. Toxicol. Appl. Pharmacol. 48: A49.

Smith, J.H., D.C. Bomberger, Jr. and D.L. Haynes. 1981. Volatilization rates of intermediate and low volatility chemicals from water. Chemosphere. 10(3): 281-291.

Spanggord, R.J., T. Mill, T.W. Chou, W.R. Mabey, J.H. Smith and S. Lee. 1980. Environmental fate studies on certain munitions wastewater constituents. Final Report, Phase I. Literature Review. SRI Project No. LSU-7934. Contract No. DAMD 17-78-C-8081. U.S. Army Medical Res. and Develop. Command, Fort Detrick, MD.

Spanggord, R.J., K.E. Mortelmanns, A.F. Griffin and V.F. Simmon. 1982. Mutagenicity in Salmonella typhimurium and structure-activity relationships of waste-water components emanating from the manufacture of trinitrotoluene. Environ. Mutagen. 4(2): 163-180.

SRI (Stanford Research Institute) International. 1984. 1984 Directory of Chemicals Producers: United States of America. Menlo Park, CA.

Tabak, H.H., C.W. Chambers and P.W. Kabler. 1964. Microbial metabolism of aromatic compounds. I. Decomposition of phenolic compounds and aromatic hydrocarbons by phenol-adapted bacteria. J. Bacteriol. 87: 910-919.

TSCA-ITC. 1984. Information Review m-Dinitrobenzene. Working Draft. Prepared under EPA Contract 68-01-6650 for TSCA Interagency Testing Committee.

U.S. EPA. 1977. Computer print-out of non-confidential production data from TSCA Inventory. U.S. EPA, OPTS, CID, Washington, DC.

U.S. EPA. 1980. Dinitrobenzenes. Health and Environmental Effects. Washington, DC.

U.S. EPA. 1983. Reportable Quantity Document for Dinitrobenzenes. Prepared for Office of Solid Waste and Emergency Response by ECAO, Cincinnati, OH.

U.S. EPA. 1985. Summary of Current Acceptable Daily Intakes (ADIs) for Oral Exposure. Internal Review Draft ECAO-CIN-432, March, 1985. Environmental Criteria and Assessment Office, Cincinnati, OH.

U.S. EPA/NIH (National Institute of Health). 1984. OHM-TADS (Oil and Hazardous Materials Technical Assistance Data System.) On-line computer.

USITC (U.S. International Trade Commission). 1981. Imports of Benzenoid Chemicals and Products 1980, 1982. USITC Publ. 1163, Washington, DC.

USITC (U.S. International Trade Commission). 1983. Imports of Benzenoid Chemicals and Products 1980, 1982. USITC Publ. 1401, Washington, DC.

USITC (U.S. International Trade Commission). 1984. Synthetic Organic Chemicals. U.S. Production and Sales, 1983. USITC Publication 1588. p. 41.

van der Schalie, W.H. 1983. Acute and Chronic Toxicity of 3,5-Dinitroaniline, 1,3-Dinitrobenzene and 1,3,5-Trinitrobenzene to Freshwater Aquatic Organisms. Army Medical Bioengineering Research and Development Lab. Report No. USAMBRDL-TR-8305. Fort Detrick, MD. NTIS ADA 138408.

Veith, G.D., K.J. Macek, S.R. Petrocelli and J. Carroll. 1980. An evaluation of using partition coefficients and water solubility to estimate bioconcentration Factors for Organic Chemicals in Fish. In: Aquatic Toxicology. ASTM STP 707. J.G. Eaton, P.R. Parrish and A.C. Hendricks, Eds., American Society for testing and Materials. p. 116-129.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals, 2nd ed. Van Nostrand Reinhold Co., New York. p. 568-569.

Wentsel, R.S., R.G. Hyde, W.E. Jones, III, M.J. Wilkinson and W.E. Hayward, III. 1979. Problem Definition Study on 1,3-Dinitrobenzene, 1,3,5-Trinitrobenzene and Di-n-Propyl Adipate. Report No. 49-5730-08. Atlantic Research Corp., Alexandria, VA. NTIS AD-A099732/0.

White, R.P. and J. Hay. 1901. Some recent inquiries and researches into the poisonous properties of naphthaline and the aromatic compounds. Lancet. 2: 582-584. (Cited in TSCA-ITC, 1984; Wentsel et al., 1979)

APPENDIX

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of:

CA SEARCH (Files 308, 309, 310, 311, 320
TOXLINE
RTECS
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
TSCAPP

These searches were conducted in December, 1984, with the exception of the TOXLINE, which was searched in February, 1984 and kindly supplied by Dynamac Corp. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1980. Documentation of the Threshold Limit Values. 4th ed. (Includes Supplemental Documentation, 1981, 1982, 1983). Cincinnati, OH. 486 p.

ACGIH (American Conference of Governmental Industrial Hygienists). 1983. TLVs: Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1983-1984. Cincinnati, OH. 94 p.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2A. John Wiley and Sons, NY. 2878 p.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2B. John Wiley and Sons, NY. p. 2879-3816.

Clayton, G.D. and F.E. Clayton, Ed. 1982. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2C. John Wiley and Sons, NY. p. 3817-5112.